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Oxidative Stress Responses in *Haemophilus parainfluenzae*

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The microbiome or bacterial composition of the oral cavity is extremely diverse. One interesting relationship is between *Haemophilus parainfluenzae* and *Streptococcus mitis*, two species that are abundant members of the human oral microbiome. Streptococcus sp. are well documented to produce substantial amounts of hydrogen peroxide in aerobic environments in the oral cavity. In fact, previous work in Dr. Matthew Ramsey's lab has shown that S. mitis-produced peroxide can kill *H. parainfluenzae* in a dose-dependent manner. However, *H. parainfluenzae* is repeatedly co-isolated with and shown to grow directly adjacent to *Streptococcus* species including *S. mitis*, suggesting that *H. parainfluenzae* possesses the capability to detoxify hydrogen peroxide.

Previous research in the Ramsey lab has shown that a transcription factor called OxyR plays an important role for the defense of *H. parainfluenzae* in the presence of hydrogen peroxide. OxyR, upon reacting with hydrogen peroxide, is activated and modulates expression of 47 genes that directly and indirectly deal with oxidative stress. This includes genes such as catalase (*katA*), cytochrome C peroxidase (*ccpd*), DNA starvation protection protein (*dps*), Glutaredoxin- peroxiredoxin (*pdgx*), Glucose-6 phosphate dehydrogenase (*g6pd*), and peroxiredoxin (*prx*).

This project investigates what gene or combination of genes controlled by OxyR are responsible for the survival of *H. parainfluenzae* when living in the presence of hydrogen peroxide produced by Streptococci. While most aerobic species rely on catalase as the main or sole contributor to peroxide resistance, the *H. parainfluenzae* response is multifactorial and relies on the contribution of multiple genes examined here in this project.